be used to follow the course of allergic alveolitis. It is much less clear, however, that the intensity of the reaction can be used to suggest active disease. In our patients, the intensity of the precipitin reaction diminished with time. Several of the tests became negative after a period of three months. In fact, there seemed to be an inverse relationship between the height of the Histoplasma mycelial titer and the presence of positive precipitin tests.

The diagnosis of acute histoplasmosis can at times be difficult. Cultures are often negative and serologic titers slow to rise. In our case, the Histoplasma mycelial titer of 1:8 was significant and pointed to the correct diagnosis. Wheat et al have recently emphasized that mycelial titers of 1:8, when accompanied by M or H bands, are highly suggestive of histoplasmosis.

Of the three recognized forms of extrinsic allergic alveolitis, none clearly fits the clinical picture demonstrated by our patients. Pulmonary mycotoxicosis, on the other hand, could cause a clinical picture similar to the one we encountered. This condition is an acute pneumonia caused by massive fungal spore inhalation. Fever and pulmonary infiltrates can last for days, thus closely mimicking acute histoplasmosis. Precipitins, however, are characteristically absent in mycotoxicosis, and, of course, CF titers for histoplasmosis would not be expected.

What then is the significance of the positive precipitin tests we have identified? It seems probable that these antibodies represent an intense, early immunologic response to fungal inhalation. Their real significance lies in the fact that they might be misinterpreted and could conceivably lead to an incorrect diagnosis and inappropriate therapy. Clinicians should be aware that such an antibody response can occur in acute histoplasmosis.

ADDENDUM

Since completion of this paper, we have studied the sera of 12 additional patients with acute histoplasmosis. Forty percent of these patients manifest strongly positive precipitins against farmer's lung antigens.

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Pulmonary Artery Catheter Occlusion as an Indication of Pulmonary Embolus

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Two patients with respiratory failure had consistently reproducible findings on insertion of flow directed pulmonary artery catheters which led to the subsequent diagnosis of pulmonary embolus. Occlusion of the catheter tip and development of a rising “overwedge” tracing on deflation of the balloon may mean the catheter tip has become embedded in clot. When combined with the inability to obtain a wedge tracing, the deflation “overwedge” tracing should alert the physician to the possibility of unsuspected pulmonary embolus.

Pulmonary embolus is a common consideration in patients with shock or acute respiratory failure, even when there is an apparent etiology to explain the illness. It is particularly difficult to make the diagnosis of pulmonary embolus in patients with pneumonia, chronic lung disease, chronic pulmonary hypertension and congestive heart failure. To evaluate and treat patients with severe respiratory illnesses or circulatory collapse, balloon flotation pulmonary arterial catheters are frequently used. We describe two patients with an unusual finding on insertion of a Swan-Ganz type catheter that led to the evaluation for, and diagnosis of, pulmonary embolus.

CASE REPORTS

CASE 1

A 79-year-old alcoholic man was admitted to the Intensive Care Unit after being found semiconscious on the floor of his home. He was unable to give any history. Initial examination showed blood pressure of 180/90 mm Hg, heart rate, 94/min; respiration rate, 24/min; and temperature, 39°C rectally. Chest examination showed bilateral diffuse rales and rhonchi, more pronounced at the bases. There was no murmur, gallop, or evidence of increased central venous pressure. Admission leukocyte count was 14,200/cu mm. The hemoglobin was 16.5 g/dl. Arterial blood gas levels were: pH, 7.47; Pco2, 30 mm Hg; Paco2, 64 mm Hg; and O2 saturation 92 percent on 4 liters of oxygen by nasal prongs. Chest roentgenogram showed cardiomegaly, a tortuous aorta and a small infiltrate in the right lower lung field. Gram stain of sputum showed numerous Gram-positive cocci. The electrocardiogram showed sinus tachycardia, an old anterior infarction, and nonspecific ST-T wave change. Computed tomographic examination of the brain, and lumbar puncture gave normal results. He was initially treated for presumed aspiration pneumonia. On the second hospital day the patient developed supraventricular tachycardia, progressive hypoventilation, and hypoxemia requiring intubation.

A flow-directed balloon flotation catheter was passed easily into...
the pulmonary artery and an adequate pulmonary arterial tracing was obtained. However, a pulmonary wedge tracing was unobtainable. On deflation of the balloon, the catheter tip became occluded resulting in an “overwedge” pressure tracing (Fig 1). This was repeatable on multiple trials leading to the hypothesis that the main pulmonary artery was partially filled with soft clot which obstructed the passage of the inflated balloon. The deflated catheter could then enter the clot and become occluded resulting in the characteristic rising pressure tracing. Pulmonary angiography revealed multiple large central pulmonary emboli. The pulmonary artery catheter tip was seen to be centrally located and apparently incorporated in clot (Fig 2).

CASE 2

A 75-year-old woman with the diagnosis of carcinoma of the rectum underwent rectosigmoidectomy ten days prior to transfer to the Intensive Care Unit. Prior to admission to the Intensive Care Unit she was found unconscious on the floor of her hospital bathroom with an unobtainable blood pressure. At that time her chest physical examination and roentgenogram were normal. Her electrocardiogram showed an old inferior wall myocardial infarction. Arterial blood gas levels on FiO₂ of 0.6 and PEEP of 5 cm H₂O were: pH, 7.53; PCO₂, 29.3 mm Hg; Pao, 96 mm Hg, and O₂ saturation, 97 percent. During the placement of a flow-directed pulmonary arterial catheter it was noted that a wedge tracing was unobtainable despite easy passage into the pulmonary artery. On deflation of the balloon an occlusive “overwedge” pressure tracing was obtained. This led to the consideration of pulmonary embolus which was diagnosed by finding multiple large defects on radionuclide perfusion scanning.

DISCUSSION

During the insertion of Swan-Ganz catheters there are many possible causes of occlusion of the catheter tip, including juxtaposition to a vessel wall, over-inflation of the balloon with herniation over the catheter tip, entanglement in cardiac trabeculations, small localized clots at the catheter tip, and kinking of the catheter. In the cases reported here the occlusion tracings were noted in a normally functioning catheter, whose frequency response and damping coefficient were known to be adequate, immediately on deflation of the balloon. No attempt had been made to advance the catheter beyond its prior easily obtainable pulmonary arterial placement. Both pulmonary arterial placements were confirmed by chest roentgenograms to be in a central, large vessel. In addition, one catheter was seen by pulmonary angiography to be incorporated in a large central clot.

A recent report stressed the significance of an inability to perform thermodilution cardiac output measurements in a patient with large central pulmonary emboli due to presumed insertion of the catheter tip into clot and obscuration of the thermistor. Neither of our patients underwent cardiac output measurements prior to pulmonary angiography. However, the diagnosis of an occluded catheter tip is easily made by observing the constantly elevating pressure tracing due to the continual addition of small quantities of fluid to the noncompliant catheter tubing by the flush device. If other causes of occlusion have been eliminated, then the more expensive and invasive testing for pulmonary emboli may be appropriate.

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